Propagation of Information in Populations of Self-Replicating Code

Johan Chu[1] and Chris Adami[2,3]

[1]*Department of Physics 114-36*
[2]*W. K. Kellogg Radiation Laboratory 106-38*
[3]*Computation and Neural Systems 139-74*
California Institute of Technology, Pasadena, CA 91125

We observe the propagation of information in a system of self-replicating strings of code ("Artificial Life") as a function of fitness and mutation rate. Comparison with theoretical predictions based on the reaction-diffusion equation shows that the response of the artificial system to fluctuations (*e.g.* velocity of the information wave as a function of relative fitness) closely follows that of natural systems. We find that the relaxation time of the system depends on the speed of propagation of information and the size of the system. This analysis offers the possibility of determining the minimal system size for observation of non-equilibrium effects at fixed mutation rate.

I. INTRODUCTION

Thermodynamic equilibrium systems respond to perturbations with waves that re-establish equilibrium. This is a general feature of statistical systems, but it can also be observed in natural populations, where the disturbance of interest is a new species with either negligible or positive fitness advantage. The new species spreads through the population at a rate dependent on its relative fitness and some basic properties of the medium which can be summarized by the diffusion coefficient. This problem has been addressed theoretically [1] and experimentally (see *e.g.* [2] and references therein) since early this century. The application of the appropriate machinery (diffusion equations) to the spatial propagation of *information* rather than species, is much more recent, and has been successful in the description of experiments with *in vitro* evolving RNA [3,4].

Systems of self-replicating information (*cf.* the replicating RNA system mentioned above) are often thought to represent the simplest living system. They offer the chance to isolate the mechanisms involved in information transfer (from environment into the genome) and propagation (throughout the population), and study them in detail.

It has long been suspected that living systems operate, in a thermodynamical sense, far away from the equilibrium state. On the molecular scale, many of the chemical reactions occurring in a cell's metabolism require non-equilibrium conditions. On a larger scale, it appears that only a system far away from equilibrium can produce the required diversity (in genome) for evolution to proceed effectively (we will comment on this below).

In the systems that we are interested in – systems of self-replicating information in a noisy and information-rich environment – the processes that work for and against equilibration of information are clearly mutation and replication. In the absence of mutation, replication leads to a uniform non-evolving state where every member of the population is identical. Mutation in the absence of replication, on the other hand, leads to maximal diversity of the population but no evolution either, as selection is absent. Thus, effective adaptation and evolution depend on a balance of these driving forces (see, *e.g.* [7,8]). The relaxation time of such a system, however, just as in thermodynamical systems, is mainly dictated by the mutation rate which plays the role of "temperature" in these systems [8]. As such, it represents a crucial parameter which determines how close the system is to "thermodynamical" equilibrium. Clearly, a relaxation time larger than the average time between (advantageous) mutations will result in a non-equilibrium system, while a smaller relaxation time leads to fast equilibration. The relaxation time may be defined as the time it takes information to spread throughout the entire system (*i.e.* travel an average distance of half the "diameter" of the population). A non-equilibrium population therefore can always be obtained (at fixed mutation rate) by increasing the size of the system. At the same time, such a large system segments into areas that effectively cannot communicate with each other, but are close to equilibrium themselves. This may be the key to genomic diversity, and possibly to speciation in the absence of niches and explicit barriers.

The advent of artificial living systems such as tierra [5,7] and avida [10,11] have opened up the possibility of checking these ideas explicitly, as the evolutionary pace in systems both close and far away from equilibrium can be investigated directly. As a foundation for such experiments, in this paper we investigate the dynamics of information propagation in the artificial life system sanda, a variant of the avida system designed to run on arbitrarily many parallel processors. This is a necessary capability for investigating arbitrarily large populations of strings of code. The purpose of our experiments is two-fold. On the one hand, we would like to "validate" our Artificial Life system by comparing our experimental results to theoretical predictions known to describe natural systems, such as waves of RNA strings replicating in $Q\beta$-replicase [3,4]. On the other hand, this benchmark allows us to determine the diffusion coefficient and veloc-

1

ity of information propagation from relative fitness and mutation rate. Finally, we arrive at an estimate of the minimum system size which guarantees that the population will not, on average, equilibrate.

In the next section we briefly describe sanda and its main design characteristics. The third section introduces the reaction-diffusion equation for a discrete system and analytical results for the wavefront velocity as a function of relative fitness and mutation rate. We describe our results in the subsequent section and close with some comments and conclusions.

II. THE ARTIFICIAL LIFE SYSTEM "SANDA"

Like avida, sanda works with a population of strings of code residing on an $M \times N$ grid with periodic boundary conditions. Each lattice point can hold at most one string. Each string consists of a sequence of instructions from a user-defined set. These instructions, which resemble modern assembly code and can be executed on a virtual CPU, are designed to allow self-replication. The set of instructions used is capable of universal computation.

Each string has its own CPU which executes its instructions in order. A string self-replicates by executing instructions which cause it to allocate memory for its child, copy its own instructions one by one into this new space, and then divide the child from itself and place it in an adjacent grid spot. The child then is provided with its own virtual CPU to execute its instructions.

When a string replicates, it places its child in one of the eight adjacent grid spots, replacing any string which may have been there. Which lattice point is chosen can be defined by the user. In our experiments, we have used both random selection and selection of the oldest string in the neighbourhood. As we shall see, the selection mechanism has a significant effect on the spread of information.

It should be noted that this birth process, and indeed all interactions between strings, are local processes in which only strings adjacent to each other on the grid may affect each other directly. This is important as it both supplies the structure needed for studies of spatial characteristics of populations of self-replicating strings of code, and allows longer relaxation times – making possible studies of the equilibration processes of such systems and their nonequilibrium behavior.

This process of self-replication is subject to mutations or errors which may lead to offspring different from the original string and in most cases non-viable (*i.e.* not capable of self-replication). Of the many possible ways to implement mutations, we have used only copy errors — every time a string copies an instruction there is a finite chance that instead of faithfully copying the instruction, it will instead write a randomly chosen one. This chance of mutation is implemented as a mutation rate R – the probability of copy-error per instruction copied. A mutation rate R for a string of length ℓ will therefore lead

to a fidelity (probability of the copied string being identical to the original) $\alpha = (1 - R)^{\ell}$. This then, allows us to evolve a very heterogeneous population from an initially homogeneous one. The resulting evolution, coevolution, speciation etc. have been and continue to be studied. [6–11]

What decides whether one particular sequence of instructions (or genotype) will increase or decrease in number are the rate at which it replicates, and the rate that it is replaced at. In our model, the latter is genotype independent (the "chemostat" regime). Accordingly, we define the former (*i.e.* its average replication rate) as the genotype's fitness. In other words, fitness is equal to the inverse of the time required to reproduce (gestation time).

To consistently define a replication rate, it is necessary to define a unit of time. Previously, in tierra and avida, time has been defined in terms of instructions executed for the whole population (scaled by the size of the population in the case of avida). In sanda, we define a physical time by stipulating that it takes a certain finite time for a cell to execute an instruction. This base execution time may vary for different instructions (but is kept constant in all experiments presented here). The *actual* time a cell takes to execute a certain instruction is then increased or decreased by changing its "efficiency". Initially, each cell is assigned an efficiency near unity, $e = (1 + \eta)$, where η represents a small stochastic component. In summary, the time it takes a cell to execute a series of instructions depends on the number of instructions, the particular instructions executed, and the cell's efficiency.

Self-replication consists of the execution of a certain series of instructions by the cell. Thus, the fitness of the cell (and its respective genotype) is just the rate at which this is accomplished and depends explicitly on the cell's efficiency. We can assign better (or worse) efficiency values to cells which contain certain instructions or which manage to carry out certain operations on their CPU register values. This allows us to influence the system's evolution so as to evolve strings which carry out allocated tasks. A cell that manages a user-defined task can be assigned a better efficiency for accomplishing it. Such cells, by virtue of their higher replication rate, would then have an evolutionary advantage over other cells and force them into extinction. At the same time, the discovery that led to the better efficiency is propagated throughout the population and effectively frozen into the genome.

In addition to the introduction of a real time, sanda differs from its predecessors in its parallel emulation algorithm. Instead of using a block time-slicing algorithm to simulate multiple virtual CPUs, sanda uses a localized queuing system which allows perfect simulation of parallelism.

Finally, sanda was written to run on both parallel processors and single processor machines. Therefore, it is possible, using parallel computers, to have very large populations of strings coevolving. This permits studies of extended spatial properties of these systems of self-

replicating strings and holds promise of allowing us to study them away from equilibrium.

III. DIFFUSION AND WAVES

Information in sanda is transported mainly by self-replication. When a string divides into an adjacent grid site, it is also transferring the information contained in its code (genome) to this site. We have looked at the mode and speed of this transfer in relation to the fitness of the genotype carrying the information, the fitness of the other genotypes near this carrier, and the mutation rate.

Consider what happens when one string of a new genotype appears in an area previously populated by other genotypes. We will make the assumption that the fitness of the other viable (self-replicating) genotypes near the carrier are approximately the same. This holds for cases where the carrier is moving into areas which are in local equilibrium. We will use f_c for the fitness of the newly introduced (carrier) genotype and f_b for the fitness of the background genotypes. If $f_c < f_b$, obviously the new genotype will not survive nor spread.

In the following, we have studied three different cases: diffusion, wave propagation, and wave propagation with mutation.

The diffusion case represents the limit where the fitness of both genotypes are the same. It turns out that this can be modelled as a classical random walk. On average, if the carrier string replicates it will be replaced before it can replicate again. This is effectively the same as the carrier string *moving* one lattice spacing in a random direction chosen from the eight available to it. The random walk is characterized by the disappearance of the mean displacement and the linear dependence on time of the mean squared displacement:

$$\langle r \rangle(t) = 0 \tag{3.1}$$
$$\langle r^2 \rangle(t) = 4Dt \tag{3.2}$$

where D is defined as the diffusion coefficient.

For our particular choice of grid and replication rules, we find for the diffusion coefficient of a genotype with fitness f,

$$D^{(b)} = \frac{3}{8}a^2 f \tag{3.3}$$

where a is the lattice spacing. This holds for a "biased" selection scheme where we select the oldest cell in the neighbourhood to be replaced. (See below.)

If $f_c > f_b$ then we find that instead of diffusion we obtain a roughly circular population wave of the new genotype spreading outward. We are interested in the speed of this wavefront.

Let us first treat the case without mutation. If the radius of this wavefront is not too small we can treat the distance from the center of the circle r as a linear

coordinate. We define $\rho(r,t)$ as the mean normalized population density of strings of the new genotype at a distance r from the center at a time t measured from our initial seeding with the new genotype. We assume that the ages of cells near each other have roughly the same distribution and that this distribution is genotype independent, ensuring that the selection of cells to be replaced does not depend on genotype either.

Then, we can write a flux equation (the reaction-diffusion equation) which determines the change in the population density $\rho(r,t)$ as a function of time

$$\frac{\partial \rho(r,t)}{\partial t} =$$

$$\left[\frac{3}{8}\rho(r-a,t) + \frac{1}{4}\rho(r,t) + \frac{3}{8}\rho(r+a,t) \right] f_c \left(1 - \rho(r,t)\right)$$

$$- \left[\frac{3}{8}(1 - \rho(r-a,t)) + \frac{1}{4}(1 - \rho(r,t)) \right.$$

$$\left. + \frac{3}{8}(1 - \rho(r+a,t)) \right] f_b \rho(r,t) . \tag{3.4}$$

Since we are interested in the speed of the very front of the wave, we can assume ρ to be small. Also, from physical considerations we assume ρ is reasonably smooth. Then, we can use a Taylor expansion for $\rho(r \pm a,t)$ and keep the lowest order terms to obtain

$$\frac{\partial \rho(r,t)}{\partial t} = \frac{3}{8} a^2 f_c \frac{\partial^2 \rho(r,t)}{\partial r^2} + (f_c - f_b)\rho(r,t) . \tag{3.5}$$

This can be solved for the linear wavefront speed $v^{(b)}$ yielding [12]

$$v^{(b)} = a \sqrt{\frac{3}{2}} \sqrt{f_c(f_c - f_b)} \tag{3.6}$$

$$= 2\sqrt{D_c^{(b)}(f_c - f_b)} \tag{3.7}$$

where $D_c^{(b)}$ is the diffusion coefficient of the carrier genotype when using a biased (by age) selection scheme.

To study the case of wave-propagation with mutation we shall make the assumption that all mutations are fatal. We can then calculate a steady state density of nonviable cells δ,

$$\delta = 1 - \alpha^{1/8} \tag{3.8}$$

where the fidelity α is the probability that a child will have the same genotype as its parent (*i.e.*, not be mutated). As mentioned earlier, the fidelity is related to the mutation rate R by

$$\alpha = (1 - R)^\ell \tag{3.9}$$

where ℓ is the length of the particular string. Modifying our previous flux equation to take into account these new factors and repeating our previous analysis gives us

$$v^{(b)} = 2\alpha\sqrt{D_c^{(b)}(f_c - \alpha^{1/8} f_b)} . \tag{3.10}$$

Let us now consider the effects of different selection schemes for choosing cells to be replaced. The relations we derive above hold true for the case in which we replace the oldest cell in the 8-cell neighbourhood when replicating ("age-based" selection). Another method of choosing a cell for replacement is to choose a random neighbouring cell regardless of age. This scheme, which we term "random selection" as opposed to the biased selection treated above, effectively halves the replication rate of all cells. It follows that the diffusion coefficient is also halved,

$$D^{(r)} = \frac{3}{16}a^2 f \qquad (3.11)$$

$$= \frac{1}{2}D^{(b)} \qquad (3.12)$$

and for the velocity of the wavefront (with no mutation) we find

$$v^{(r)} = 2\sqrt{D_c^{(r)}\frac{(f_c - f_b)}{2}} \ . \qquad (3.13)$$

In Fig.1, we show a histogram of the number of offspring that a cell obtains before being replaced by a neighbour's offspring, for the biased selection case (left panel) and the random case (right panel). As expected from general arguments, half of the cells in the random selection scenario are replaced before having had a chance to produce their first offspring (resulting in a reduced diffusion coefficient), while biased selection ensures that most cells have exactly one child.

IV. RESULTS

We carry out our experiments by first populating the grid with a single (background) genotype of fitness f_b. Then, a single string of the carrier genotype with fitness f_c is placed onto a point of the grid at time $t = 0$. We then observe the position and speed of the wavefronts formed, the mean squared displacement of the population of carrier genotypes, and various other parameters as a function of time.

With f_b kept constant[1], we have varied f_b/f_c from 0.1 to 1.0 in increments of 0.1. Also, the mutation rate R was varied from 0 to 14×10^{-3} mutations per instruction, in increments of 1×10^{-3}.

A comparison of the theoretical *vs.* measured mean square displacement as a function of time for a genotype with no fitness advantage compared to its neighbours ($f_b/f_c = 1$) is shown in Fig.2. The data were obtained from approximately 1500 runs. The solid lines represent the (smoothed) averages of our measurements (for biased

---

[1]The gestation time was approximately 330,000, where the base execution time for each instruction was (arbitrarily) set to 1000: $f_b = \frac{1}{330000}$

and random selection schemes), while the dashed lines are the theoretical predictions obtained from the diffusion coefficients (3.3) and (3.11) respectively. The slopes of the measured and predicted lines agree very well confirming the validity of our random walk model and the diffusion coefficient predicted by it (without any free parameters). The slight discrepancy between the experimental curves and the predicted ones at small times is due to a finite-size effect that can be traced back to the coarseness of the grid.

Fig.3 shows the measured values of the wavefront speed for cases where $f_c > f_b$ and without mutation, with the corresponding predictions. Again, the higher curve is for biased and the lower for random selection. Note that the wavefront speed gain from an increase in fitness ratio is much better than linear. Note also that all predictions are again free of *any* adjustable parameters.

The dependence of this curve on the mutation rate is shown in Fig.4. Increasing the mutation rate tends to push the speed of the wave down. It should be noted, however, that because we have only used copy mutations there is no absolute cutoff point or error threshold α_c where all genotypes cease to be viable, with $\alpha_c > 0$. Rather, genotypes can spread until α is very close to the limit $\alpha_c = 0$.

Finally, we plot the dependence of the wavefront speed on the mutation rate for a fixed value of the fitness ratio ($f_b/f_c = 0.6$) in Fig.5. Data were obtained from an average of four runs per point in the biased selection scheme. Again, the prediction based on the reaction-diffusion equation with mutation agrees well (within error bars) with our measurements.

V. DISCUSSION AND CONCLUSIONS

Information propagation via replication into physically adjacent sites can be succinctly described by a reaction-diffusion equation. Such a description has been used in the description of *in-vitro* evolution of RNA replicating in Qβ-replicase [3,4], as well as the replication of viruses in a host environment [13]. The same equation is used to describe the wave behavior of different strains of *E. Coli* bacteria propagating in a petri dish [14], even though the means of propagation in this case is motility rather than replication.

We have constructed an artificial living system (sanda) based on the avida design which allows the investigation of large populations of self-replicating strings of code, and the observation of non-equilibrium effects. The propagation of information was observed for a broad spectrum of relative fitness, ranging from the diffusion regime where the fitnesses are the same through regimes where the difference in fitness led to sharply defined wavefronts propagating at constant speed. The dynamics of information propagation led to the determination of a crucial time scale of the system which represents the average time for
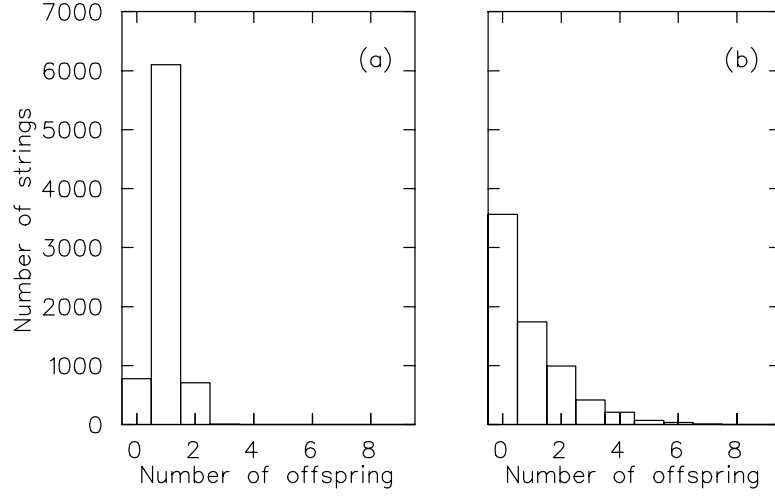
FIG. 1 Distribution of number of strings generating different numbers of offspring, for the biased selection case [panel (a)] and the random selection scenario (b).
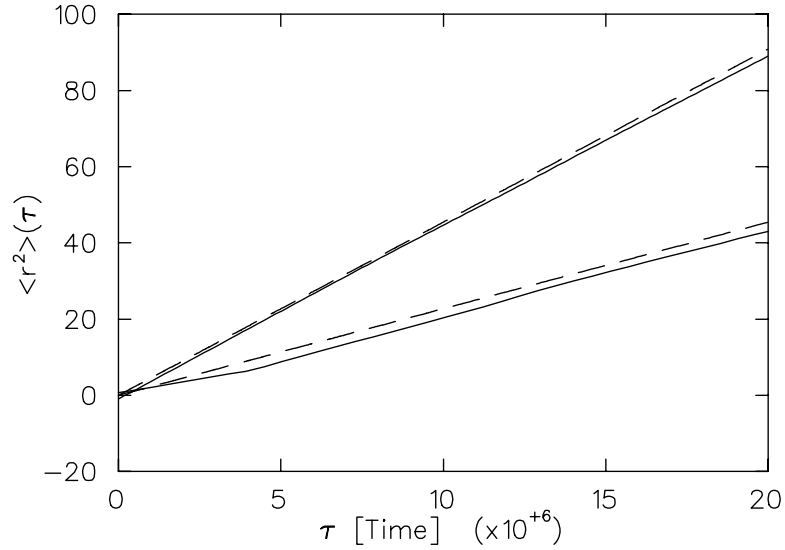

FIG. 2 Mean squared displacement of genome as a function of time due to diffusion. Solid lines represent experimental results obtained from 1500 independent runs. Dashed lines are theoretical predictions. The upper curves are obtained with the biased selection scheme while the lower curves result from the random selection scenario.
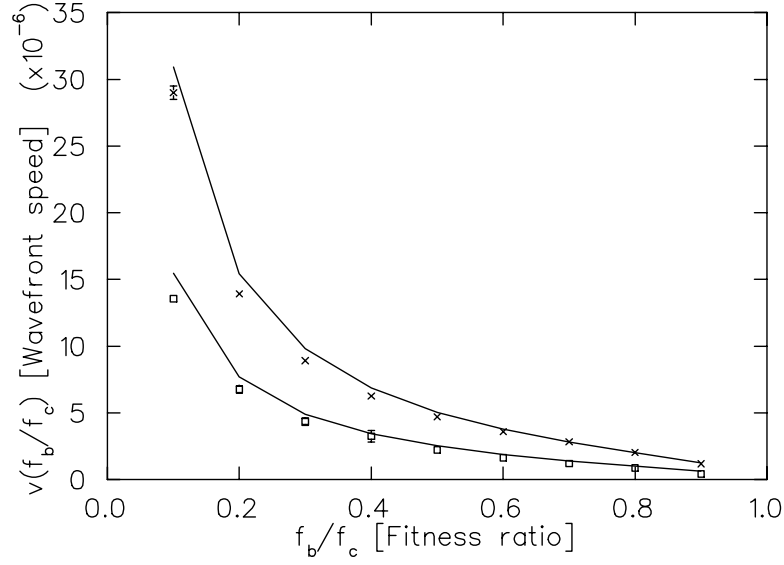
FIG. 3 Wavefront speed of a genotype with fitness f_c propagating through a background of genotypes with fitness f_b, averaged over four runs for each data point. Upper curve: biased selection, lower curve: random selection. Solid lines are predictions of Eqs. (3.7) and (3.13).
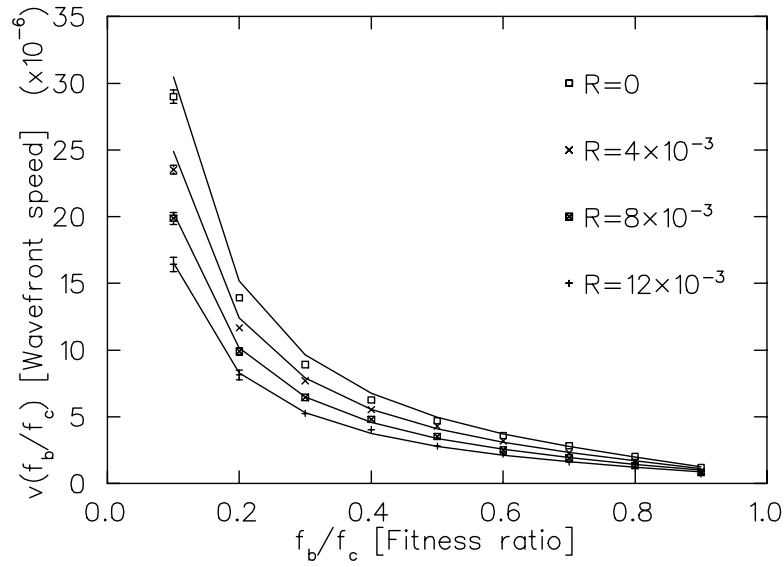

FIG. 4 Measured wavefront speeds versus fitness ratio for selected mutation rates R (symbols) are plotted with the theoretical predictions from Eq. (3.10) (for the biased selection scheme only).
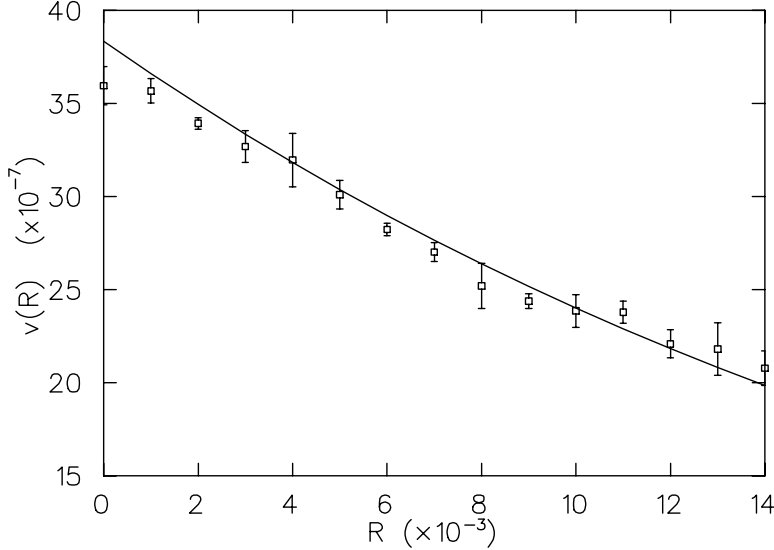
FIG. 5 Wavefront speed of a genotype (biased selection) with relative fitness $f_b/f_c = 0.6$ as a function of mutation rate (symbols). Solid line is prediction of Eq. (3.10).

the system to return to an equilibrium state after a perturbation. This relaxation time depends primarily on the size of the system, and the speed of information propagation within it. Equilibration can only be achieved if the mean time between (non-lethal) mutations is larger than the mean relaxation time. Thus, a *sufficiently* large system will never be in equilibrium. Rather, it is inexorably driven far from equilibrium by persistent mutation pressure.

For artificial living systems such as the one we have investigated, it is possible to formulate an approximate condition which ensures that it will (on average) never equilibrate, but rather consist of regions of local equilibrium that never come into informational contact. From the timescales mentioned above, we determine that the number of cells N in such a system must exceed a critical value:

$$N > \left(\frac{2\, v(f)}{R_\star\, a} \right)^{2/3} , \qquad (5.1)$$

where $R_\star$ is the rate of *non-lethal mutations*, $v(f)$ the velocity of information waves, and a the lattice spacing (assuming a mean time between non-lethal mutations $t_\star \approx (N\, R_\star)^{-1}$).

Beyond the obvious advantages of a non-equilibrium regime for genomic diversity and the origin of species, such circumstances offer the fascinating opportunity to investigate the possibility of non-equilibrium pattern formation in (artificial) living systems. However, the most interesting avenue of investigation opened up by such artificial systems is that of the study of the fundamental characteristics of life itself. Since it is widely believed that many of the processes that define life, including evolution, occur in a state which is far from equilibrium, to

study such processes it is necessary to have systems which exhibit the properties of life we are interested in and that can be quantitatively studied in a rigorous manner in this regime. The availability of artificial living systems as experimental testbeds that can be scaled up to arbitrary population sizes on massively parallel computers is a step in this direction.

[1] R.A. Fisher, Ann. Eugen **7** (1937)355.
[2] Th. Dobzhansky and S. Wright, Genetics **28** (1943)304.
[3] G.J. Bauer, J.S. McCaskill, and H. Otten, Proc. Natl. Acad. Sci. USA **86** (1989)7937.
[4] J.S. McCaskill and G.J. Bauer, Proc. Natl. Acad. Sci. USA **90** (1993)4191.
[5] T. S. Ray, in *Artificial Life II:* Proceedings of an Interdisciplinary Workshop on the Synthesis and Simulation of Living Systems, Santa Fe Institute Studies in the Sciences

of Complexity, Proc. Vol. 10, edited by C. G. Langton et al., Addison-Wesley, Reading, MA, p. 371 (1992).

[6] T. S. Ray, Physica **D 75** (1994)239; Artificial Life **1** (1994)195.

[7] C. Adami, Physica **D 80** (1995)154.

[8] C. Adami, Artificial Life **1** (1994)429.

[9] C. Adami, Phys. Lett. **A 203** (1995)23.

[10] C. Adami and C.T. Brown, In R.A. Brook and P. Maes (Eds.), *Artificial Life IV*: Proceedings of the Fourth International Workshop on the Synthesis and Simulation of Living Systems, p. 377. MIT Press, Cambridge, MA (1994).

[11] C. Adami, C.T. Brown, and M. Haggerty, Proc. of 3rd Europ. Conf. on Artificial Life, June 4-6, 1995, Granada, Spain, Lecture Notes in Computer Science p.503, Springer Verlag (1995).

[12] M. C. Cross and P.C. Hohenberg, Rev. Mod. Phys. **65** (1993)851.

[13] J. Yin and J.S. McCaskill, Biophys. J. **61** (1992)1540.

[14] K. Agladze et al., Proc. Roy. Soc. Lond. B, **253** (1993)131.